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CLINICAL ASPECTS OF EXPERIMENTAL INFECTION OF HORSES WITH THE VIRUS OF AUJESZKY'S DISEASE

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The problem of the clinical infection of horses injected with the virus of Aujeszky's disease has been poorly covered in the literature up to now.

There is contradictory data on this question. Some investigators consider that horses are in general not susceptible to artificial infection or to infection in natural conditions, while others (Lukashov, Pastegayeva) maintain that horses are susceptible to artificial intracerebral infection.

Having observed cases of experimental infection of horses by the virus of Aujeszky's disease, we thought we should publish our clinical data.

For the test we used young, healthy, well-fed horses from the ages of 3½ to 7½ years, which were checked to be sure they were free of parasitic blood diseases and infectious diseases in general, including infectious anemia. All the horses were quarantined for 3 months before the test and after the test were kept in isolation.

For infection of the horses we used a biologically checked, typical virus of Aujeszky's disease in the form of a tissue emulsion of the brain, spleen, and lymph glands of rabbits which had died of Aujeszky's disease, in a 1:5 physiological solution. The horses were infected by a 5-ml dose subcutaneously in the upper third of the neck.

Thirty-three percent of the infected horses became ill. Among the group of diseased horses, 63 percent were blinded and 50 percent died. Observing the clinical aspects of the diseased horses, we noted that the course of the disease had two basic forms; violent, with attacks of great excitement, and mild.

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Of the seven horses which died, three had a violent form and four a mild form of the disease.

For characterization of these forms of the clinical course of the disease, we cite entries from the case-history book.

A reddish-colored gelding, 5 years old, was injected with the virus of Aujeszky's disease in a 40-ml dose, 7 grams of brain. On the fourth day after the injection, an increase in temperature up to the limits of 39.2 - 39.7° was noted. However, the general condition of the animal was unchanged, and the appetite was normal.

On 23 December 1940, temperature in the morning was 39.5, in the evening 40.0°. The appetite was sluggish. The mucus of the oral cavity and the conjunctiva of the eyes were hyperemic with a somewhat yellowish hue. Physiological functions were without any significant deviations.

On 24 December, temperature in the morning was 38.5, in the evening 39.7°. General condition was without change.

On 26 December, temperature in the morning was 38.2, in the evening 39.4°. The condition was unchanged.

On 27 December, temperature in the morning was 38.9°. The morning feed was only half eaten. Acute sluggishness changed into oppression. The horse stood downcast, with its head hanging into the manger, periodically making nervous movements of the lips, with defecation stopped, and peristaltic action of the intestines absent. In 15 hours, agitation was noted. The animal seemed alert and frequently perked up its ears. There was a chewing motion with gritting of the teeth and a fibrillar muscular tremor, spreading into the chest, shoulder blades, and crupper.

After 2 hours, the horse made an abrupt movement of its back, dashed forward suddenly, striking its chest on the manger, then made a number of running motions, and, lurching forward, threw its forelegs into the manger, stretched its head up, and pressed its nose against the wall, thus shutting its nostrils and interfering with its breathing.

These phenomena increased in scope. In addition to exhibiting the fibrillary muscular tremors and the chewing action, the animal made grasping motions with the lips, and exhibited a great deal of salivary flow and perspiration. The horse remained wet. It did not respond to shouts or other external stimuli. The animal could not be led from the stall. Acute excitement lasted for 20-25 minutes and then the animal quickly subsided into a comatose state. The horse dropped with exhaustion, and lay with outstretched legs and the head thrown back, indifferent to its surroundings. Coronary and tendon reflexes were inhibited, and the respiration was slow, 5-7/min. The temperature increased slightly (38.5°). The comatose condition lasted for 25-30 minutes, after which it changed to strong excitement. The excitement increased, and passed into a frenzy, which was continued until complete exhaustion of the animal. Then the horse lay in an unconscious state, making feeble running motions with its legs. Within 24 hours, with complete decline of its strength, the animal died.

In the dying horse, the periods when the violent state alternated with the depressed state lasted 9 hours. In other horses with this same clinical course of the disease, death occurred after 8-10 hours. In cases of violent form of the course of Aujeszky's disease, we were unable to find one case which recovered.

Of the 11 horses afflicted by the mild form of the disease, four died.

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A 3½-year-old mare was infected by the virus in a dose of 5 ml (1 gram of brain). On the ninth day after infection, 12 December 1949, the animal showed an increase in temperature up to 40.8° in the morning and 40.0° in the evening. At the same time, there appeared asthenia, unsteady gait, and slack, uncertain movements.

On 13 December, temperature in the morning was 40.8°, in the evening 41.0°. In addition to the same symptoms, the animal abstained almost entirely from food, and hyperemia of the conjunctiva of both eyes was noted.

On 14 December, temperature in the morning was 40.4°, in the evening 39.5°. The symptoms were the same.

On 15 December, the temperature in the morning was 38.4°, in the evening 38.5°. The animal was in a depressed condition and its appetite was sluggish. The animal stood in a downcast manner, holding hay which it had taken in its lips for 1-2 hours, and then sluggishly and reluctantly chewing it. The respiration was slow, 8-9/min, and the pulse was slow as well, 26-28 beats/min. The heart tones were muffled.

On 16 December, temperature in the morning was 38.4°, in the evening 39.0°. The condition of the animal was the same.

On 17 December, temperature in the morning was 40.3°, in the evening 39.7°. Besides the symptoms indicated above, in the morning there was noted a twitching of the eyelid and lip, a gritting of the teeth, and atonia of the intestines.

On 18 December, temperature in the morning was 38.9°, in the evening 39.1°. The symptoms were the same.

On 19 December, temperature in the morning was 39.2°, in the evening 40.2°. The condition of the animal deteriorated, strong depression was noted, the urine changed to a color resembling that of linseed oil and became viscous and sticky in consistency.

On 20 December, temperature in the morning was 39.9°, in the evening 39.7°. The animal was strongly depressed. Cardial activity was weak, and as a consequence, there was cyanosis of the oral cavity and the nose. Also noted were tremors of the lips and eyelids, muscular fibrillary tremor, and complete refusal of food. The depression was alternated with short periods of spasms and shudders. There was ataxia.

On 21 December, temperature in the morning was 38.0°, in the evening 38.3°. The same symptoms, with increased salivation. The sensitivity of the skin increased. Hyperemia and swelling of the conjunctiva were noted. There was fibrillary spasmodic twitching of the muscles. The animal frequently lay down and got up again.

During 13 hours the indicated symptoms changed and the animal became weaker. The animal lay down and was unable to rise. After complete exhaustion, the animal died at 1700 hours on 22 December.

The remaining three horses died with about the same clinical course of the disease.

Our investigations confirmed the greater susceptibility of young horses, 3½-5½ years old, to artificial infection by the virus of Aujeszky's disease, in comparison with older horses, 8-10 years old. Our investigations also confirmed the fact that increasing the magnitude of the doses of virus material and also virulence makes possible the generation of the disease.

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Pathological and Anatomical Changes

The corpses of the horses which died from the violent form of Aujeszky's disease had abrasions on the skin of their dewlaps, kneecaps of their front legs, eye sockets, cheek bones, and the front part of their heads, and their lips were swollen and the front parts of the gums were bruised. On sectioning, the subcutaneous cellular tissue was found to be swollen and to have a yellowish tinge, with the icterus more pronounced in the corpses of horses which did not have the violent form of the disease. The smooth membrane of the cerebrum and medulla was swollen and the vessels were congested. The vascular plexus of the cerebrum was filled with blood. In the corpses of the horses which died with the violent form of the disease, the hemispheres of the cerebrum, the oblongata, and the cerebellum had foci of the infection, expressed by inflammation of the vessels and leakage of blood.

The muscles on sectioning were found to be flabby and dull in appearance, like boiled meat. The heart was flabby and the walls were stretched, with clots of fibrin in the ventricles. There was venous congestion in all the internal organs. The lungs were swollen and on sectioning dark viscous uncoagulated blood drained from them, confirming the presence of asphyxia. The stomach and the small intestine were empty, and in two cases undigested hay was found in the stomach. The mucous membrane of the stomach was greatly inflammed and thickened, particularly the membrane of the bottom of the stomach, where there was hemorrhagic inflammation and formation of cherry-colored ulcers. There were analogous changes at the bottom of the bladder. The small intestine was filled with gas and the mucous membrane in it exhibited a diffused rosy shade in spots. The large intestine was filled with a dense fecal mass covered with a layer of mucus. The mucous membrane of the large intestine was thickened and hyperemic, with hemorrhage in spots. The mesenteric lymphatic glands were enlarged and juicy. Bacteriological cultures were made from all the dead horses' hearts, livers, and spleens. All the cultures were found to be sterile.

To confirm that the cause of death was Aujeszky's disease, in two cases, we intramuscularly infected rabbits with a brain emulsion from a horse which had died of the severe form. The rabbits usually died within 4-5 days with typical symptoms of Aujeszky's disease.

Treatment

From the first days of the illness, a specific therapeutic serum against Aujeszky's disease was administered to the horse in large doses, as much as 150 ml, intravenously and subcutaneously. In some horses the doses were increased until up to 2 liters had been given, with no appreciable effect. In all probability, the serum against Aujeszky's disease, as all antivirus serums, exerted a weak therapeutic effect, notwithstanding its favorable prophylactic properties.

Starting with the first day of the illness, symptomatic treatment was also administered. The horses were given caffeine, urotropine, mucilaginous concoctions, and Glauber's and Carlsbad salts in the established doses, together with administration of glucose and physiological solution, artificial feeding, including nutrient enemas, and cleaning out the rectum by means of laxative enemas. Symptomatic treatment allowed us to save 50 percent of the sick horses.

On the basis of the clinical manifestations of Aujeszky's disease and the pathological and anatomical picture on examination of the dead horses, it is possible to assert that the course and pathological changes in this disease have much in common with the clinical manifestations of equine encephalomyelitis. Because of this circumstance, it is possible to assume that in farms where Aujeszky's disease has made its appearance among swine and cattle, it has also infected horses. However, in those cases, it has been registered as virus equine encephalomyelitis.

M. Ya. Ivannikov, Doctor of Veterinary Medicine, stated that this was the case in 1939 on farms where Aujeszky's disease was present among swine and cattle. At the same time, it infected five horses, three of which died. He registered these cases as encephalomyelitis.

Professor P. S. Solomkin confirmed that there were analogous (rare) cases of complex infections at farms where Aujeszky's disease was identified in an animal experiment, using rabbits.

In the light of this data, we recommend, in all cases of the disease on farms where various animals have Aujeszky's disease and where there are horses with encephalomyelitis, that biological tests be applied on rabbits as a method of excluding the possibility of Aujeszky's disease or confirming its presence. For the tests, small pieces should be taken from the brain (from the hemispheres of the brain, the medulla oblongata, and the cerebellum) and used to infect rabbits in the form of a tissue suspension in dilutions of 1:50 and doses of 0.2 to 0.5 ml.

Conclusions

1. A considerable percentage of horses was infected by means of experimental introduction of the virus of Aujeszky's disease.

2. The course of the disease took either severe or mild forms.

3. Infection of horses by Aujeszky's disease in the majority of cases resulted in loss of vision.

4. Young horses $3\frac{1}{2}$-$5\frac{1}{2}$ years old have a tendency toward more severe infection.

5. The clinical aspects and pathological and anatomical changes in cases of Aujeszky's disease in horses are very similar to those occurring in equine encephalomyelitis, so that it is necessary to take this factor into account in establishing the diagnosis, particularly on farms where this disease is present among other animals.

6. We did not observe scratching of the lip in the sick horses, although this had been reported by a series of authors.

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